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Case Report

Central nervous system tuberculoma mimicking a brain tumor: A case report☆,☆☆

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ABSTRACT

The central nervous system (CNS) is a rare but serious site of tuberculosis spread that manifests in three forms: meningitis, spinal arachnoiditis, and CNS tuberculoma. CNS tuberculoma, or intracranial tuberculous granuloma, is a caseating or non-caseating granulomatous reaction within the brain parenchyma that may mimic a brain tumor. We present the case of a 10-year-old male patient with a travel history to Western Africa who presented to our institution after his fourth tonic-clonic seizure over 2 months. MRI of the brain revealed a solitary cortical/subcortical enhancing intracranial mass with intralesional hemorrhage and mineralization, pathologically proven to represent a CNS tuberculoma. While rare, this etiology should be considered with the appropriate travel history and for which prompt treatment may improve outcomes in the pediatric population.

Introduction

Central nervous system (CNS) tuberculosis (TB) is a rare but devastating sequela of *Mycobacterium tuberculosis* infection. Only 1%-5% of TB patients will develop CNS complications, but the mortality rate ranges from 15% to 40%. TB spread to the CNS presents in 3 forms: meningitis, spinal arachnoiditis, and CNS tuberculoma [1]. Meningitis is the most prevalent presentation in countries with low TB incidence like the United States, but the three are equally common in countries with high TB incidence. A CNS tuberculoma consists of

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conglomerate granulomatous foci in the CNS contained by the host's immune system. Patients are often present with neurological symptoms (eg, headache, seizure, hemiplegia) without indications of systemic infection or meningitis. CNS tuberculomas may involve various structures in the CNS, including the meninges, ventricular system, pituitary gland, and elsewhere [1]. The appearance of CNS tuberculomas on imaging studies may mimic other pathological processes, posing a challenge to clinicians when patients present primarily with neurological symptoms and without other typical presenting symptoms of a TB infection (eg, cough, hemoptysis, weight loss). Here, we report a case of a 10-year-old patient who suffered 4 tonic-clonic seizures over the course of 2 months and whose MRI demonstrated a cortical/subcortical mass considered to be most consistent with a primary neoplasm. He underwent craniotomy and excision of the mass, and a diagnosis of CNS tuberculoma was rendered.

Case report

A 10-year-old previously healthy male presented with his fourth tonic-clonic seizure in 2 months. After his second seizure, he was prescribed Levetiracetam 100 milligrams BID and had 2 subsequent breakthrough seizures. His vitals were normal and neurological examination in the emergency department identified weakness in his right leg. Social history revealed family ties to Western Africa. He had last traveled to Guinea and the Ivory Coast 2 years prior, and his mother had last visited 1 year prior.

MRI of the brain revealed a 1.8 cm cortical/subcortical enhancing mass within the left precuneus with significant adjacent vasogenic edema (Fig. 1). The mass demonstrated areas of intrinsic T1 shortening and associated susceptibility hypointensity with predominant negative phase shift and

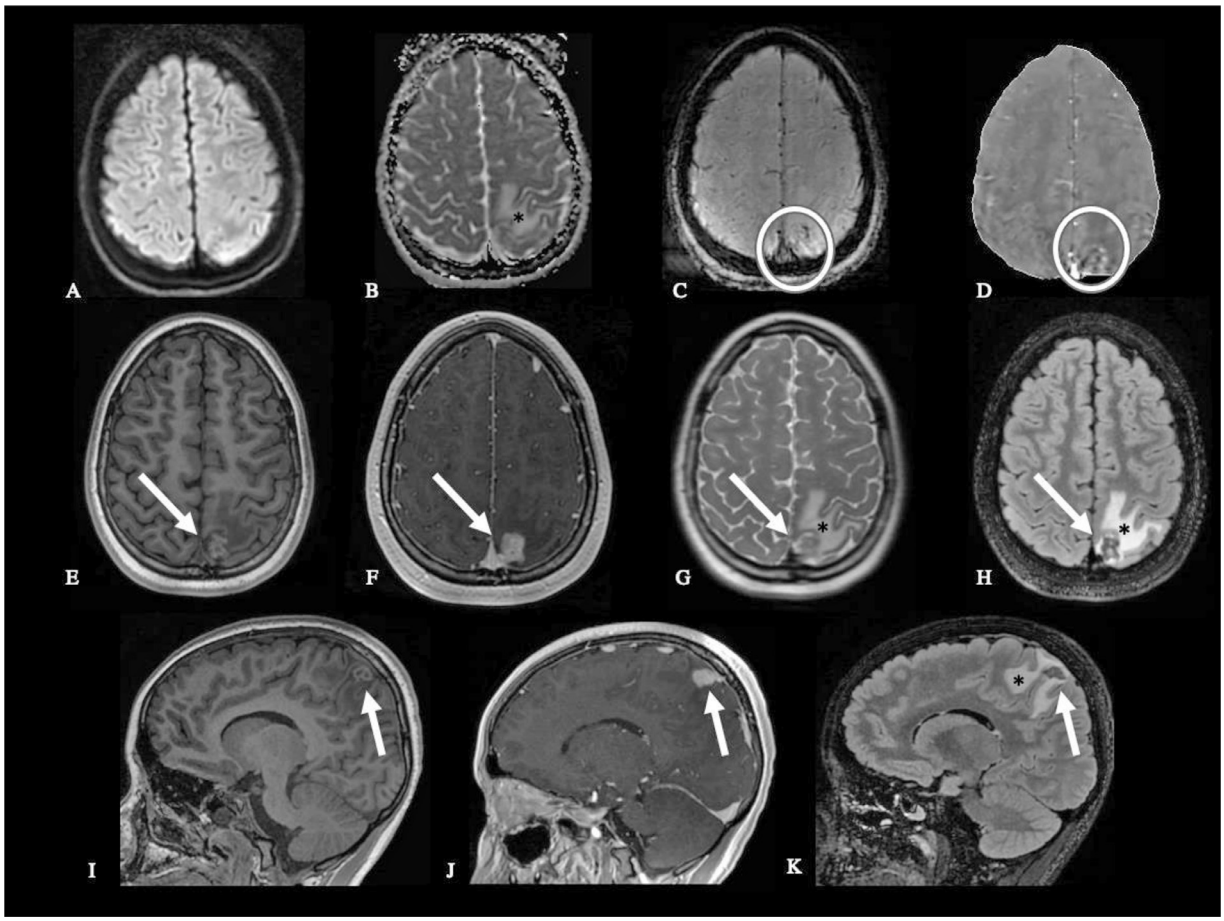

Fig. 1 – Axial diffusion-weighted imaging (DWI) (A), apparent diffusion coefficient (ADC) map (B), susceptibility-weighted imaging [SWI] (C), quantitative susceptibility map [QSM] (D), axial T1-weighted (E), post-contrast axial T1-weighted (F), axial T2-weighted (G), axial T2-weighted fat-saturated fluid-attenuated inversion recovery (FLAIR) (H), sagittal T1-weighted (I), sagittal postcontrast T1-weighted (J) and sagittal T2 fat-saturated FLAIR (K) sequences demonstrate a 1.8 cm enhancing cortical/subcortical mass (white arrow; E–J) centered within the left posteromedial precuneus with adjacent significant vasogenic edema (black asterisk; B, G, H, K). Mass demonstrates foci of susceptibility with both positive and negative phase shift (white circle; C and D), suggesting mineralization and intralesional blood products.

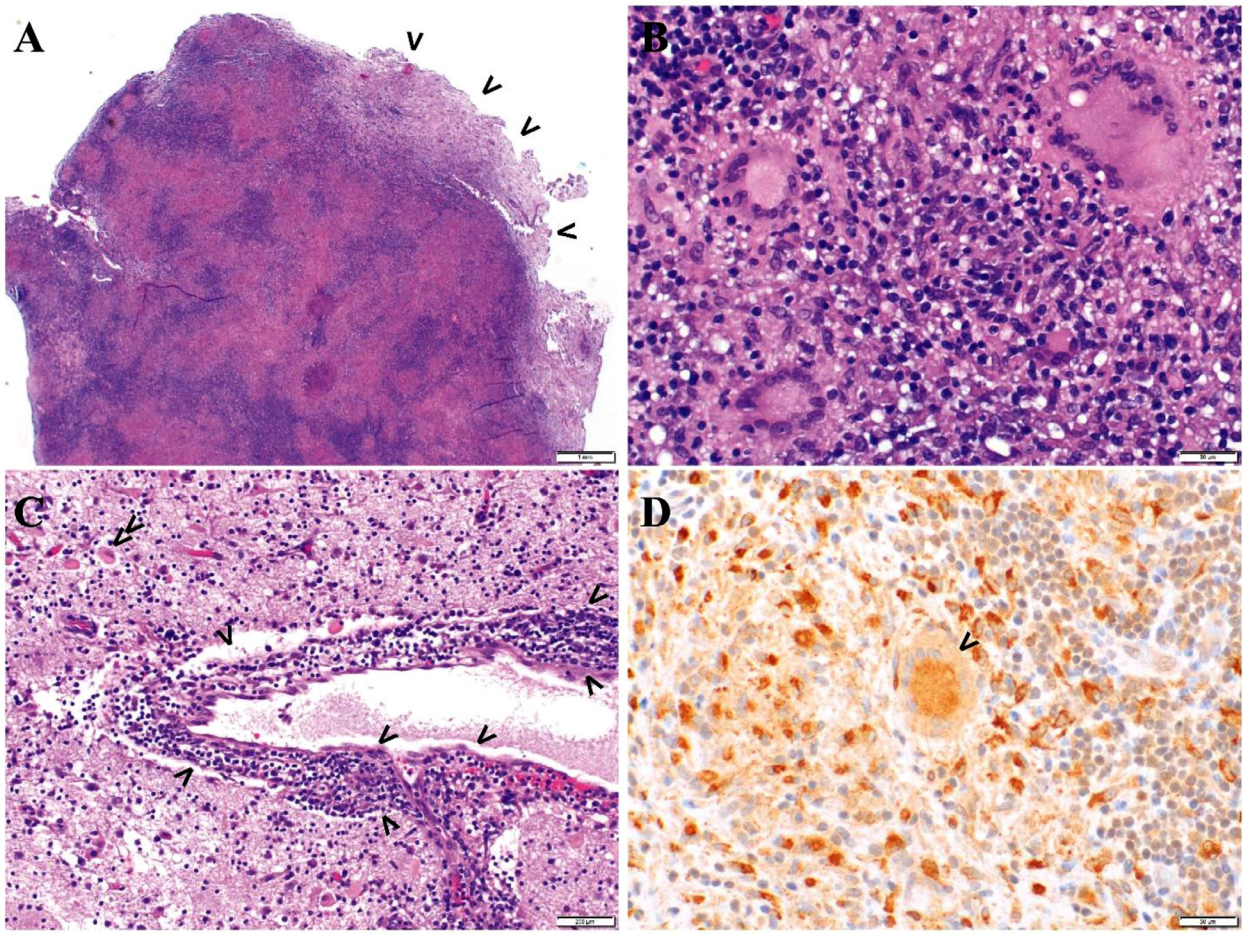

Fig. 2 – Histopathological analysis. Low power photomicrograph of the excised lesion shows a rind of reactive brain parenchyma (arrows) surrounding a large focus of back-to-back nodular granulomas (A). Higher power shows a robust chronic inflammatory infiltrate comprising lymphocytes, macrophages, and multinucleated giant cells, characteristic of granulomas. The vast majority in this case were noncaseating. Some foci of the lesion demonstrated chronic inflammation within the leptomeninges and surrounding large caliber vessels (arrows, C). KP1 (CD68) immunohistochemical staining highlights macrophages in brown with the cytoplasm of multinucleated cells also showing labeling (arrow, D). (A–C) Hematoxylin and eosin stain. (D) CD68 immunostain. Scale bars: A = 1 mm; B and D = 50 um; C = 200 um.

few foci of positive phase shift, suggesting both mineralization and intralesional blood products. Differential considerations for this mass included cortically-based neoplasms, such as oligodendroglioma, pleomorphic xanthoastrocytoma, and ganglioglioma, as well as infectious or inflammatory etiologies.

Given the patient's concerning clinical course and imaging findings, he underwent a left parietal craniotomy with complete excision of the mass, without complication. On the day of his operation, the patient tested positive on the QuantiFERON-TB test, heightening suspicion of a CNS tuberculoma. The specimen revealed extensive intraparenchymal granulomatous inflammation (Figs. 2A and B) with associated reactive gliosis and leptomeningeal chronic inflammation (Fig. 2C), consistent with a diagnosis of CNS tuberculoma. Immunostaining for KP1 (CD68) highlighted the robust macrophage infiltrate as well as multinucleated giant cells characteristic of granulomatous inflammation (Fig. 2D). While only a rare form suspicious for acid-fast bacillus was seen on a Fite Faraco special stain (not shown), PCR testing of the formalin-fixed paraffin embedded lesional tissue block confirmed the presence of *M. tuberculosis* in the specimen.

The postoperative course consisted of antituberculosis treatment with isoniazid, rifampin, pyrazinamide, ethambutol, and Vitamin B6.

Discussion

Disseminated spread to the CNS is seen in only 1%-5% of patients with TB but is responsible for disproportionate morbidity and mortality. Tuberculosis infection of the CNS may manifest as meningitis, spinal arachnoiditis, or CNS tuberculoma. A tuberculoma is an aggregate of granulomatous tissue in the CNS that has been contained by the host's immune

system. While typically solitary, patients with immunocompromised states may present with multiple lesions. In regions endemic to TB, CNS tuberculomas comprise 5%-10% of intracranial masses, but only 0.2% in Western countries [1].

CNS tuberculomas may present with focal neurological deficits from the growing intracranial mass without any of the symptoms typically associated with a TB infection, and only 30% of CNS tuberculoma patients have a positive chest radiograph [2]. The imaging findings of a CNS tuberculoma are varied and often non-specific, adding to the diagnostic challenge.

This patient's clinical presentation – seizure – parallels that classically seen in patients with brain tumors. Seizure is the most common presenting symptom for oligodendrogliomas and gangliogliomas, and the second most common presenting symptom for pleomorphic xanthoastrocytomas. Despite the focal lesion, the seizure presentation is typically generalized and tonic-clonic, as seen in this patient [3].

The imaging features of a CNS tuberculoma depend on the stage of the lesion. A noncaseating granuloma is typically hypointense to surrounding cerebral parenchyma on T1-weighted images, hyperintense on T2-weighted images, and demonstrates homogenous postcontrast enhancement. Caseating granulomas with a dense center appear hypointense or isointense to gray matter on T1-weighted images and isointense on T2-weighted images. Upon administration of gadolinium, caseating granulomas typically show a ring-enhancing "target sign" that is pathognomonic for a CNS tuberculoma [2]. The lesion, in this case, was isointense to the surrounding brain parenchyma on T2-weighted images, in contrast to oligodendrogliomas, gangliomas, and pleomorphic xanthoastrocytomas, which are typically T2 hyperintense [4–6].

Diagnostic proof of a TB infection is not required to initiate anti-TB treatment in patients with high clinical suspicion, and prognosis is heavily influenced by the stage of the disease course when treatment is started. Prompt delivery of the 4-drug regimen of isoniazid, rifampin, pyrazinamide, and ethambutol can alleviate the neurological symptoms of the CNS tuberculoma and mitigate the need for surgery [1]. While systemic complications of craniotomies occur less often in the pediatric population than in adults, the operation is not without risks. A comprehensive analysis revealed an increased risk of postoperative new-onset neurological defects, local and systemic complications, and a 2.0% surgical mortality rate [7].

Clinicians should consider CNS tuberculomas in their differential diagnosis for solitary intracranial mass lesions, particularly when provided the appropriate travel history. The diagnosis can be challenging, especially when the patient does not present with typical TB symptoms in a non-endemic part of the world. For clinicians practicing in an area with low TB incidence, a thorough family and travel history are critical for identifying TB as a potential source of their patient's disease course.

Patient consent

Written, informed consent was acquired from the patient's legal guardian before the submission of this manuscript.

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